be at least 150% above threshold to achieve good results with unilateral ECT (2).

Some patients who did not respond to unilateral ECT were switched to bilateral ECT. However, the authors do not mention whether every patient who did not respond to unilateral ECT was switched. If this was not done, the results could be skewed.

One wonders what prevented the authors from independently measuring the outcome for patients receiving unilateral and bilateral treatments. This would resolve the debate on whether the poor outcome was due to the type of treatment used. The authors chose instead to quote another study that was done on a biased sample (3).

To hypothesize that ECT and heterocyclic antidepressants have similar mechanisms of action is highly speculative, especially after acknowledging in the same article that heterocyclics alone are not an effective treatment for psychotic depression, whereas ECT is!

REFERENCES

1. Prudic J, Haskett RF, Mulsant B, Malone KM, Pettinati HM, Stephens S, Greenberg R, Rifas SL, Sackeim HA: Resistance to antidepressant medications and short-term clinical response to ECT. Am J Psychiatry 1996; 153:985–992
2. Sackeim HA, Prudic J, Devanand DP, Kiersky JE, Fitzsimons L, Moody BJ, McElhiney MC, Coleman EA, Settembrino JM: Effects of stimulus intensity and electrode placement on the efficacy and cognitive effects of electroconvulsive therapy. N Engl J Med 1993; 328:839–846
3. Prudic J, Sackeim HA, Devanand DP: Medication resistance and clinical response to electroconvulsive therapy. Psychiatry Res 1990; 31:287–296

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Drs. Prudic and Sackeim Reply

TO THE EDITOR: We thank Dr. Chandragiri for commenting on our study. Contrary to his interpretation of our previous work, there is no evidence regarding the efficacy of right unilateral ECT administered with a stimulus intensity 50% above seizure threshold (1). We have only demonstrated that right unilateral ECT is ineffective when administered just above seizure threshold. Nonetheless, only five of the 100 patients in our study received right unilateral ECT at 50% above seizure threshold, and all patients who did not respond to this form of ECT (N=3) were crossed over to bilateral ECT at 150% above seizure threshold. Of the remaining patients in the study who were initially treated with right unilateral ECT, the *minimum* dose was 150% above the initial seizure threshold.

The key finding in this study was the multisite confirmation that resistance to antidepressant medications during the index episode predicted clinical outcome with ECT in primary, unipolar, nonpsychotic patients. In Dr. Chandragiri's second and third points, the issue is raised as to whether the findings were contingent on a large number of patients who were being treated with right unilateral ECT and not bilateral ECT. In our study, 90 of the 100 patients were treated initially with right unilateral ECT. Thirty-two of the 45 patients (71%) who initially did not respond to right unilateral ECT were crossed over to bilateral ECT. Consequently, the bulk of patients who did not respond to right unilateral ECT subsequently received bilateral ECT. In a 1990 study that Dr. Chandragiri cited, we had already shown that medication resistance predicted a lower response rate to bilateral ECT. Our new study indicates that this effect pertains to both right unilateral and bilateral ECT. Indeed, we conducted a new logistic regression that predicted initial ECT response or nonresponse on the basis of categorization as medication resistant and whether the patient was treated with only right unilateral ECT (N=58) or received bilateral ECT as an initial or crossover modality (N=42). Medication resistance was strongly related to ECT short-term outcome (χ^2=7.57, df=1, p=0.006), while ECT modality was not (χ^2=1.07, df=1, p=0.30).

The argument that ECT and antidepressants have independent mechanisms of action had been supported by the previously untested belief that ECT is as effective in patients who are antidepressant resistant as in patients who have not failed an adequate medication trial. This argument is now more tenuous given our findings that antidepressant-resistant patients have a poorer response to ECT. Since we also found that resistance to heterocyclic antidepressants predicted a poorer response to ECT, whereas resistance to selective serotonin reuptake inhibitors (SSRIs) did not, it is reasonable to speculate that there is more overlap in mechanisms among heterocyclics and ECT than for SSRIs and ECT in the treatment of nonpsychotic depression. The efficacy of ECT in the treatment of psychotic depression is not germane.

REFERENCE

1. Sackeim HA, Decina P, Kanzler M, Kerr B, Malitz S: Effects of electrode placement on the efficacy of titrated, low-dose ECT. Am J Psychiatry 1987; 144:1449–1455

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Reprints of letters to the Editor are not available.

Corrections

In "Correlation Between Reduced in Vivo Benzodiazepine Receptor Binding and Severity of Psychotic Symptoms in Schizophrenia" by Geraldo F. Busatto, M.D., Ph.D., et al. (January 1997, pp. 56–63), the fourth line of the footnote (page 56) should indicate that the paper is also from the Institute of Nuclear Medicine, University College and Middlesex School of Medicine, London.

In "Posttraumatic Stress Disorder Associated With Peacekeeping Duty in Somalia for U.S. Military Personnel" by Brett T. Litz, Ph.D., et al. (February 1997, pp. 178–184), the sentence beginning on the 10th line of the last paragraph on page 179 should read: "On average, the survey was completed 14.9 weeks (SD=9.2, range=62) after participants had returned to the United States."